vomiting—all symptoms disappearing in the course of two or three days. In the two cases upon which autopsy was performed, fibrinous exudates were found upon the pia mater, and serous exudate in the pericardium. A few interesting facts were brought out by the bacteriological investigations. A spheroidal coccus was obtained, sometimes isolated, sometimes in pairs, always extra-cellular, and not taking Gram's stain. This grew both aerobically and anaerobically, was found not only in the exudate, but in blood from the heart and finger, and in the tissue of the spleen. It was non-pathogenic for rabbits or white mice. Growths were obtained upon potato and bouillon, thus differing from the meningococcus of Weichselbaum. No case of direct contagion was observed. One of the nurses in the hospital, however, contracted the disease. The most benign cases were found among young infants. SAILER.

94. ZUR FRAGE DER VERÄNDERUNGEN IN DEN PERIPHERISCHEN NERVEN BEI DER CHRONISCHEN ERKRANKUNG DER GEFÄSSE DER EXTREMITÄTEN (Concerning the Changes in the Peripheral Nerves in Chronic Disease of the Vessels of the Extremities). Michael Lapinsky (Deutsche Zeitschrift für Nervenheilkunde, 13, 1898, p. 468).

Lapinsky has examined eight cases in which the arteries of one or both lower extremities were diseased, and gangrene of the limbs followed. The clinical evidences of neuritis were absent; motion and sensation were about normal. The vasa nervorum were altered, and the connective tissue of the nerves was much increased in amount, especially toward the foot, and this increase was proportionate to the change in the vessels. The nerves contained numerous spaces, supposed to be due to stagnation of the lymph. The nerve fibers were not greatly altered, except that where the endoneurium was much thickened the nerve fibers were small. These changes of the nerves were regarded as the result of vascular disease. SPILLER.

95. NERVOUS VOMITING. Bertram Hunt (Clinical Journal, Vol. XII, 1898, p. 239).

The author reports what he believes to be a fatal case of functional emesis. A married woman of 29 years had had, a year before, an attack of abdominal pain and vomiting which lasted several days and then left her entirely. Nine months after this she had a severe "bilious attack," and on the following day vomited a teacupful of bright blood. From this day to the time of admission she constantly vomited directly after meals, and also between meals, there being occasionally a small quantity of blood in the vomit. When admitted the patient was but slightly emaciated; the abdomen was rigid and tender all over, but she referred the spontaneous pain entirely to one point in the epigastrium, pointing to the painful spot with one finger in the way described in the text-books as typical of gastric ulcer. The treatment consisted of cutting off all food by the mouth, and feeding entirely by nutrient enemata; fomentations and poultices to abdomen; morphia hypodermically; while tincture of iodine, ipecacuanha wine, and tincture of opium in small and frequent doses were all tried successively by the mouth. In spite of treatment the vomiting continued almost incessantly for five days, being so continuous as to almost prevent sleep; the vomit contained bile, and occasionally small quantities of dark blood. The general nature of the case with the history of hematemesis, of course, strongly suggested gastric ulcer; but as the patient was extremely emotional, and the vomiting was unusually frequent, and did not respond to treatment applicable to gastric ulcer, it was suggested that there was a marked nervous element to be dealt with. The patient was accordingly put on drachm doses of potassium bromide by the rectum four times a day, and in thirty-six hours the vomiting, which was previously increasing in severity, had entirely ceased. In four days more she was successfully taking solid food by the mouth, and in a fortnight was discharged cured.

She remained in good health a year, when the pain and vomiting suddenly returned, and she was re-admitted on January 28, 1898. The symptoms were exactly the same as in the previous year; the vomiting being almost incessant, the vomit consisting of clear, bile-stained liquid, frequently containing traces of blood. In this attack, however, the treatment by rectal feeding, bromide and morphia was absolutely inoperative. The vomiting increased, the pulse became rapid and feeble, and in twelve days the patient died, the end being accelerated by an attack of uncontrollable diarrhea with offensive stools.

The necropsy revealed absolutely no lesion except a catarrhal inflammation of the lower end of the ileum, which, no doubt, accounted for the terminal diarrhea. The stomach was perfectly healthy, except for a few submucous hemorrhages. There was no ulceration, erosion, or atrophy of the mucous membrane. The liver, kidneys and other organs were all healthy.

It is not stated that the nervous system was examined, and apparently no microscopic examination was made at all. PATRICK.

96. EIN EXPERIMENTELLER BEITRAG ZUR FRAGE DER PERIPHEREN DEGENERATIVEN NEURITIS BEI TUBERCULOSE (An Experimental Contribution to the Question of Degenerative Peripheral Neuritis in Tuberculous Persons). Carl Hammer (Deutsche Zeitschrift für Nervenheilkunde, 12, 1898, p. 215).

Neuritis has been so frequently found in persons suffering from tuberculosis that there must be some relation between the two diseases. Hammer found that he was not able to produce neuritis in guinea pigs, in every case, by the injection of cultures of the tubercle bacilli. When the material for inoculation was taken from a tuberculous human peritoneum the results were very different, and the nerves were found markedly degenerated. The material from the human peritoneum seems, therefore, to be more virulent. He found, also, that tuberculosis in guinea pigs, produced by any form of inoculation, invariably causes changes in the spinal motor cells of guinea pigs, detectable eight days after the inoculation, and in some cases sufficiently intense to cause death of the cells. Hammer believes that the neuritis found in man in tuberculosis and other infectious diseases is probably secondary to changes in the cells. SPILLER.

97. UEBER DAS WESEN UND DIE ENTSTEHUNG DER HEMIPLEGISCHEN CONTRACTUR (Concerning the Nature and Origin of Hemiplegic Contracture). Ludwig Mann (Monatsschrift für Psychiatrie und Neurologie, Vol. IV, Nos. 1 and 2, pp. 45 and 123).

The theories which are used to explain contracture by the loss of the inhibition transmitted through the pyramidal tract, or by the stimulation of the cells of the anterior horns from secondary degeneration of these tracts, are very unsatisfactory. In hemiplegia the paralysis and contracture affect distinct groups of muscles. The muscles